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THE HOSPITAL BULLETIN

OF THE UNIVERSITY OF MARYLAND

BALTIMORE MEDICAL COLLEGE NEWS

VOL. IX MAY 15, 1913 No. 3

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Prescription Optician .. ..

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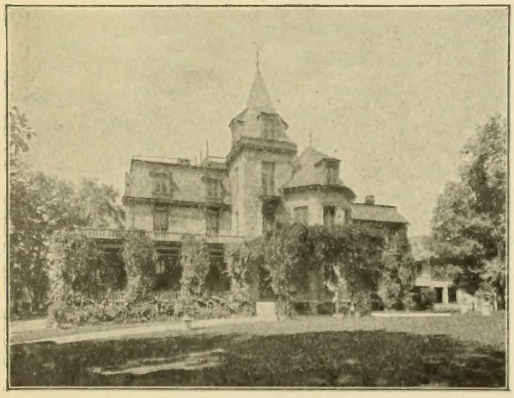

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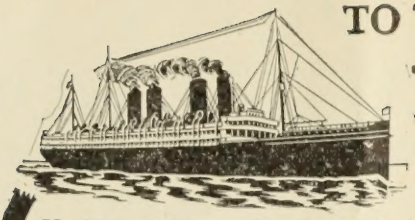

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SYMPTOMS AND PATHOLOGY OF ECLAMPSIA.

By J. M. Buch,
Senior Medical Student.

Eclampsia is an acute toxemia occurring in the pregnant, parturient or puerperal woman, and is usually characterized by clonic and tonic convulsions, during which there is a loss of consciousness, followed by more or less prolonged coma (Williams).

The word eclampsia comes from the Greek word *eclampo,* meaning *to flash out,* and refers to a sudden onset. This, however, is not the case, and, as will be mentioned later, prodromal symptoms usually occur.

The disease is seen about once in every 300 or 500 cases of pregnancy, and occurs most frequently during labor (intrapartum eclampsia); next in frequency before labor (ante-partum eclampsia, and least frequently after labor (post-partum eclampsia or puerperal eclampsia). It is seldom seen during the first half of pregnancy, increasing in frequency as term approaches.

Primiparity, hydramnios, heredity and twin pregnancies are all predisposing factors, its occurrence in the latter being tenfold that of normal.

Eclampsia is sometimes the termination of pre-eclamptic toxemia. According to Prentiss (*American Journal of Obstetrics* and *Diseases of Women and Children,* Vol. 64, page 255), by this "is meant the formation and retention within the body during pregnancy of unknown poisons that give rise to the symptoms which often precede eclampsia."

The eclamptic attack is most frequently introduced by premonitory symptoms which may last from a few hours to several days. Nausea may be present, sometimes very marked, and may or may not be associated with the ingestion of food; pain in the epigastrium, right hypochondrium or over the clavicle, especially the first, when persistent and severe, is indicative of an oncoming attack; severe frontal, parietal and rarely occipital headache; disturbances of vision, as asthenopia, amblyopia, diplopia and muscae volitantes; stupor, insomnia, mental excitement, vertigo, tinnitus aurium, sensation of weakness and edema may exist. The urine is usually scanty, containing albumen, casts and a diminished amount of urea. After such a train of symptoms the conditions may improve and the patient fall asleep, or more commonly they are followed by a typical attack which in a small proportion of cases is ushered in by a warning aura. The patient may utter a long cry or may cover her face with her hand as if to protect it from something.

A typical attack may be divided into three stages—*invasion, convulsions* and *coma.* During the *invasion* consciousness is lost, the pupils are usually dilated and the eyes stare; twitchings of the lips are followed by spastic contractions of the mouth, the face is cyanotic, the eyes roll up, and the neck rotates to the side. This gradually merges into the stage of *tonic and clonic convulsions.* In this stage the distortions of the face take on a descending character, the neck is thrown back in opisthotonus with the trunk, the fingers grasp the thumb; the muscles of respiration being affected, breathing may be suppressed or one or two breath movements may occur at the height of the convulsions; the face is congested and the lower extremities may at times contract; the tongue may partially protrude and a clear or bloody foam may come from the mouth; the contractions now may become clonic and breathing stertorous, and after a few seconds to few minutes the patient passes into the third stage or coma. The *coma* may last about half an hour or more, after which consciousness slowly returns, or the patient falls asleep and usually awakes con-

fused. The number of convulsions may vary from one to as many as 100 in severe cases, the intervals between varying from half an hour to a few seconds, one convulsion overlapping the other and giving rise to a true *status convulsivus* or *status eclampticus*, in which the patient may die without recovering consciousness, or it may partially reoccur just before the patient dies from edema of the lungs, congestion of the brain, hemorrhage, exhaustion, or later of puerperal infection.

Although the convulsions described occur with a high degree of frequency, several cases of eclampsia without convulsions have been reported.

In ante-partum eclampsia convulsions generally bring on labor, or pregnancy is artificially terminated. If labor does not result, the mother may die, or the fetus may succumb or survive the attacks, and on a second or third convulsion may be born either rigid or convulsive, and may show some of the pathologic lesions of eclampsia.

In intrapartum eclampsia labor may be rapid and convulsions may cease, or it may be delayed and cause death of the mother.

In puerperal eclampsia rapid recovery may follow after a single convulsion, or convulsions may increase in frequency soon after labor, causing a rapid death.

According to Lichtenstein (*American Journal of Obstetrics and Diseases of Women and Children*, Vol. 65, page 319), 90 per cent. of the cases of puerperal eclampsia deliver spontaneously with little loss of blood; in eclampsia the usual loss of blood is 500 c. c., or 100 c. c. to 150 c. c. more than normally, and in operative deliveries the normal loss of blood is increased by 40 per cent.; therefore, the amount of blood lost is a very important factor in the cause of puerperal eclampsia, for, acting like venesection, it relieves the hypertension and decreases the toxemia.

The *temperature* may be normal, but more commonly rises to 104° to 105° F. in the height of the attack, and gradually comes down as the patient recovers, or it may reach a much higher level just before death. Different causes have been ascribed to the rising of temperature, all of which are mere theories. The susceptibility, however, of the eclamptic patient to infection must not be disregarded in considering the etiology of the fever.

The *pulse* is full and bounding when the attack begins; it later becomes feeble and frequent, and more natural during coma. In the severe cases, however, it increases in rapidity and diminishes in strength, becoming thready and compressible as the fatal end approaches.

The *breathing* may be suppressed during the stage of convulsions, then it becomes stertorous, and gradually approaches normal as coma supervenes.

The *blood pressure* of the non-pregnant woman has been estimated to oscillate about 112 mm. of Hg.; that of the pregnant woman is usually about 118 mm. of Hg., but oscillations of 30 may occur with little significance, if any. In eclampsia with convulsions it may be as high as 200 mm. and as low as 155 mm., and convulsions do not occur when the blood pressure becomes lower as a result of poor resistance, as seen in fulminant cases or when lowered by veratrum viride (Bailey).

This hypertension is probably responsible for some of the symptoms, and in conjunction with increased permeability of the vessel walls may cause *generalized edema*, occurring sometimes in the form of "occult edema," which can only be detected by the rapid increase in weight out of all proportion to the amount or quality of food ingested.

Edema has been considered a valuable prognostic sign on the assumption that it carries away the poison, and so tends to relieve the brain of its mischievous effects.

Localized edema is usually the result of obstruction to the circulation.

The *urine* is very much decreased in amount, sometimes complete anuria occurring. Blood hyaline, granular and epithelial casts and kidney cells are all common. According to Dr. Williams, albumen is almost constant, fluctuating between 1 per cent. and 3 per cent.; is composed of serum-albumen and serum-globulin, and later disappearing. Still others claim that in some cases of puerperal eclampsia convulsions have occurred immediately after a urinary examination which did not reveal either casts or albumen.

The total nitrogen and urea decrease, while the amido-acids, creatinine and uric acid increase.

The ammonia coefficient decreases with the onset of convulsions, then rapidly rises and stays up for a variable length of time, which fact is considered by Williams to be a favorable sign.

Zinsser claims that the chlorides diminish rap-

idly toward the end in fatal cases—a tendency not noted in those that recovered.

With convalescence the urine gradually comes to normal; the nitrogenous contents increase partly from increased katabolism during involution of the uterus; the urea gradually disappears, and if persistent may be considered as due to a pre-existing chronic nephritis.

Mental symptoms may develop which may result in puerperal insanity.

Hemianopsia from cerebral lesions, albuminuric retinitis and toxic amaurosis may result.

Rarely jaundice may occur, and constitutes a bad prognostic sign.

The symptomatology may vary to some extent, and usually two clinical types are recognized—the hepatic and the nephritic types.

In the *hepatic* type the convulsions are more marked, more severe and more frequent; there is vomiting of a dark brown material; tympanites and abdominal tenderness exist; the urinary changes and edema are milder; jaundice is more common, and the prognosis more serious.

In the *nephritic* type the salient symptoms are headache, anuria, hypertension, edema, albumen, casts and hematuria. The prognosis is less grave.

PATHOLOGY.

After the discovery of albumen in the urine of eclamptic patients by Rayer in 1839, and its proof by Lever in 1843, the attention of the obstetrician was referred to the kidneys as being responsible for the symptoms of the disease, and they were supposed to be the seat of its pathognomonic lesions, but a careful consideration of post-mortem findings and their probable relationship to the prominent and fatal symptoms will disprove this old conception.

Carefully collected statistics show that the kidneys are very frequently involved in this affection, the main changes being usually of three types—*i. e.*, degenerative, acute inflammatory and chronic inflammatory. Eclampsia is generally considered to be an acute toxemia; therefore, changes of long standing or chronic inflammations of the kidneys are not very apt to be found unless they existed previous to the onset of the disease, or at least they are not to be considered as immediately fatal changes. Of all the lesions of the kidneys those most apt to be encountered in connection with acute intoxications of any kind are degenerations mainly of the parenchymatous and fatty nature and acute inflammations, such degenerations being undoubtedly of the mildest type, the more serious and lasting amyloid and hyaline degenerations being generally encountered in conjunction with chronic diseases and suppurations, especially of bones. From this simple reasoning, and admitting the acute and toxic character of the disease in question, we can justly infer that the simpler lesions of the kidneys are the ones to be expected in such a condition as this. It is true that such inference already presupposes the kidney lesions as merely secondary to toxemia, but even so post-mortem examinations and other means at our command do not prove the contrary, while both agree as to the final results, these facts being corroborated by the following statistics of Prutz:

Of 368 cases of eclampsia seven did not show any lesions in the kidneys, 46 per cent. and 11.6 per cent. showed acute and chronic nephritis, respectively, while degenerations were more frequently found. More commonly still, we find a combination of acute nephritis, with degeneration of the renal epithelium which may go on to necrosis. Other observers claim that the kidneys are very frequently found in a normal condition, and that when lesions are present they are usually secondary. So that, in conclusion, we may say that lesions in the kidneys, although often present, are not sufficiently marked to be considered pathognomonic.

THE LIVER.—Discarding the kidneys for a while, and remembering the marked decrease in the amount of urea eliminated through them by eclamptic patients, it is natural that our attention will be fixed on the liver.

Macroscopic Changes.—Deviations from the normal size and shape of the liver in eclampsia are neither characteristic nor constant, while the color of both external and cut surfaces is very much interrupted by irregularly shaped and sized spots of a reddish or yellowish color which give the organ a mottled appearance. Again, its contour may rarely be disturbed by the presence of hematomata resulting from subcapsular hemorrhages.

Microscopic Changes.—Hofbauer believes that fatty degeneration of the central portion of the lobules of the liver is found in all pregnant women the same as others sustain the occurrence of similar infiltrations in the kidneys known as

"the kidney of pregnancy," both of which subside soon after the completion of labor. The persistence or aggravation of such hepatic lesions is considered by some to be the cause of pernicious vomiting of pregnancy, while among others Des Ribes and Saint Blaise claim that they are practically the same as those of eclampsia and have the same anatomical basis.

Correct ideas as to the microscopic changes in the liver were first enunciated by Jurgens and Klebs in 1886, who considered it as a *hemorrhagic hepatitis*.

According to modern ideas, it seems to start with agglutination of the red blood cells in the smaller branches of the portal vessels, leading to thrombosis, deficient blood supply, degeneration and necrosis of the parenchyma around such areas, *i. e., at the periphery of the hepatic lobules*.

These changes are now considered to be the pathognomonic lesions of eclampsia, constituting, according to French ideas, a primary *hepato-toxemia*, taking place in probably all cases of eclampsia, and enabling us to distinguish it from pernicious vomiting of pregnancy in which the more widespread necrosis has its beginning around the central vessels of the lobule. This obviously has a very important bearing in some medico-legal cases.

Among rarer changes in connection with the liver might be mentioned emboli of hepatic cells occurring in other viscera.

The Brain.—Edema of the brain probably exists more commonly than is found in post-mortem examinations, perhaps due to a rapid subsidence after death. Many of the nervous symptoms have been variously attributed to pressure caused by edema of the brain increased by the strain of labor pains, and which have been termed "glaucoma of the brain."

Congestion is at times encountered, which, with the general hypertension generally found, may give rise to hemorrhage and apoplexy. These hemorrhages may be variously located as in the cortex, central ganglia or meninges, and in one case reported by Schmid hemorrhage in the fourth ventricle, with destruction of the vital centers, was the immediate cause of death.

Anemia and thrombosis has also been found.

Microscopic thrombi in the smaller cerebral vessels may cause small areas of necrosis.

Flattening of the convolutions is encountered.

Among the remote effects we may mention the puerperal hemianopsia caused by persistent brain lesions. The albuminuric retinitis sometimes found is probably secondary to kidney lesions.

The Heart. — Parenchymatous degeneration secondary to the toxemia is the usual finding, but thrombosis of the terminal branches of the coronary, with subsequent areas of localized necrosis and fatty degeneration, may also exist. These may give rise to acute dilatation, heart failure and death.

The wall of the smaller blood vessels and capillaries, accompanied by the increased blood pressure, may become more permeable to the fluid elements of the blood, causing edema, although some think the viscosity of the blood is increased.

The Lungs.—Here we have the same secondary changes already mentioned in other organs, as congestion and edema, this being a common cause of death. Anatomical peculiarities, however, may give rise to other changes, so that hemorrhagic infarcts may be found; aerogenic infection may cause broncho-pneumonia, and the interposition of these organs between the venous and arterial circulations, with their small capillaries, may cause them to be the first place of arrest of detached giant cells of placental origin.

In a post-mortem recently made in this hospital, besides all the changes already mentioned, a marked bloody effusion in the various serous cavities was encountered.

The foregoing constitute the main lesions of eclampsia in which the liver occupies the most important place, but other organs may be more or less implicated and have been made responsible for the cause of the disease.

For instance, the uterus and tubes may show interstitial lesions (Massen). The secretory portions of the mammary glands, although not claimed to be the seat of important changes, have been supposed to liberate some product which causes eclampsia the same as happens in the milk-fever of cows.

The thyroid gland, which normally enlarges and hypersecretes during pregnancy, giving rise sometimes to slight symptoms of hyperthyroidism, as the mild exophthalmos designated by the poet as "the soft eyes of motherhood," may fail to do so, and autopsy may reveal atrophy.

The parathyroids may show more or less indefinite changes, and Silvestry has claimed that

hypocalcification of the brain subsequent to hypoparathyroidism may be the cause of convulsions.

The spleen and adrenals rarely show indefinite changes.

Finally, the placenta may be the seat of infarcts, or hemorrhages probably predisposes by its normal fragility, influenced by the high blood pressure.

SYMPTOMATOLOGY OF GASTRIC AND DUODENAL ULCERS.

By Fuller Nance,
Senior Medical Student.

Gastric and duodenal ulcers are due to the action of the gastric juice on the stomach tissues.

The stomach is thought to be provided with a protective antienzyme which prevents the gastric juice attacking it, but by some perverted metabolism or lowered resistance through some cause its action is inhibited and we have ulcer formation.

There are four forms of peptic ulcer: (1) The common form, which has pronounced and well-recognized symptoms, or "gastrolgic dyspeptic form;" (2) latent form, which may go on for years, producing very little or no disturbance; (3) the acute perforating, which may be so rapid that it will produce perforation and death, with scarcely no local symptoms; (4) the acute hemorrhagic form, in which one of the first symptoms seen is profuse hemorrhage.

Physical examination shows very little, the diagnosis depending more on the history of the case.

In gastric ulcer, one of the first symptoms is an unusual gnawing hunger and pain in the epigastrium, which is usually relieved temporarily by the taking of food; but as the gastric juice is poured out to digest the food the pain is increased, due to the exposure of the fine terminal nerve filaments being irritated by the acid gastric juice. Some authorities claim that the free hydrochloric acid is greatly increased, while others claim that this is not true, but at some stage it seems to be in excess. The epigastric pains usually radiate toward the back, and there is great tenderness in this region on pressure, accompanied by more or less muscular rigidity on one side. Often about two hours after food the patient vomits and may suffer hemorrhage, but usually does not. The patient is treated for dyspepsia, which usually exists with a great deal of flatulency.

Blood more often exists in the stools which may be in such minute quantities as to be only detected by microscopic examination, or by testing for occult blood by Guaiac or Aloin test. Even when we find blood in stools, it does not prove peptic ulcer, as the blood may come from any portion of tract from mouth to anus. We also have to be very careful about the food that is taken before the test, as some meat or something might entirely destroy its value. An X-ray picture taken six hours after a bismuth test meal has been introduced that shows remains of bismuth, helps to confirm our diagnosis. In 100 cases operated on in one clinic, 77 cases were in males and 23 in women. When occurring in women, they seem to be in those who are addicted to tight lacing, or to have suffered with menstrual disorders.

There are about 10 gastric ulcers to one duodenal, and about 70 to 80 per cent. occur in men. They may be single or multiple, and may be associated with ulcer of stomach. They are formed in that part of the duodenum above the opening of the bileduct, where it is acted upon by the gastric juice, and is frequently associated with jaundice and sometimes with chronic Bright's disease.

The pain is longer in coming on after food, being four or five hours. The patient often wakes up at night with pain, due to late digestion of the supper, and will soon observe and tell you that a glass of milk or some food gives him relief. The pain is farther to the right, in the epigastric region or possibly in the right hypochondriac. The symptoms of pain and vomiting are not so marked.

Vomiting is not so common, and when it does occur it does not have the effect of relieving the pain. Bleeding is more common, although we seldom get it in vomit or stomach washings. It occurs in about one-third of cases and stool will often be found as a dark, sticky mass.

The patient is often very anemic from the loss of blood. It is estimated that about one-half of the cases of cancer of the stomach is preceeded by gastric ulcer.

There is nearly always associated with these

ulcers of the stomach and duodenum, eructation of gas from stomach which is of a peculiar, sour, bitter taste. There is more or less obstruction to passage of food, due to constriction of fibers of the musculature of the tissues, thereby causing the test bismuth meal to be held in the stomach longer than is normal. In a certain percentage of all cases of this character, there is tumor; i.e., a mass can be felt by the hand in palpation. This is not in all cases, but is occasionally found.

To sum up our symtoms in this condition we have: Acidity of stomach contents to a greater or lesser degree; pain of more or less severity, and coming on after food sooner or later, depending on the location of the ulcer; hemorrhage, to be found in nearly all cases if we diligently search for it; nausea and vomiting, nearly always seen at some stage; eruption of gas of peculiar sour, bitter taste; obstruction to some degree in all cases; tumor, in small percentage of cases.

UMBILICAL HERNIA.

By C. D. Whelchel,
Senior Medical Student.

Definition.—It is a protrusion of peritorium—containing at times, or permanently containing, any viscus or part of a viscus from the abdominal cavity—which is in the umbilical region, either acquired or congenital. The classes of umbilical herniae are clinically, reducible, irreducible, incarcerated, inflamed and strangulated.

Reducible—When the contents of the sac can be reduced into the abdominal cavity.

Irreducible—When the contents can't be reduced into the abdominal cavity, due to adhesions, the large size of the mass in some cases, or because of the excessive growth of omental fat.

Incarcerated—Is when obstruction takes place, due to damming back of feces or undigested food, the peristaltic wave arrested. This form is most common in irreducible hernias and during the existence of constipation.

Inflamed—Inflammation of an umbilical hernia is a local peritonitis, due to injury of the incarcerable form.

Strangulated—This is a condition in which, if the sac contains bowel, not only is the fecal circulation arrested and gas prevented from passing, but the circulation of blood in the bowel and omentum arrested. If not promptly relieved, gangrene soon occurs. The strangulation is much more dangerous if bowel is in the sac than if omentum alone.

Etiology.—Umbilical hernias may be either congenital or acquired.

Congenital—Here the sac is present at birth and due to developmental defect.

Acquired—Is due to intra-abdominal pressure. The sac is composed of a body, neck and mouth. When the sac is once formed, it usually persists throughout life, because it adheres to the surrounding structures. In this case, the contents of the sac may or may not be reducible, while the sac is irreducible. Fat may be deposited within the abdominal wall which lessens the size of the cavity, hence causing increased abdominal pressure, and the fat may protrude at the umbilicus. The mass of fat adheres to and makes traction on the peritoneum, which is drawn up to form the sac. This mode of formation is frequent in umbilical hernia.

Any condition which weakens the abdominal wall predisposes to hernia. The common cause is repeated muscular effort, which increases intra-abdominal pressure, as lifting heavy weights or any strain which causes a rise of intra-abdominal tension. An umbilical hernia may appear gradually or suddenly, depending upon the cause.

The sac of an acquired hernia exists for a longer or shorter period of time before the hernia enters it, or the sac may exist for years and the viscera never enter it.

Symptoms—In traumatic hernia the symptoms are: There may be violent pains in the mass, collapse, nausea and vomiting; also inability to walk without pain. In most cases, the viscera gradually find their way into the sac and here the pain is slight and ofttimes absent. However, at times the pain is severe. The contents may or may not be forced back into the abdominal cavity, depending upon the kind of hernia.

If the hernia be strangulated, the symptoms are: When the strangulation begins, the patient is seized with violent pains which are first intermittent, later becoming continuous. The hernia is found to be irreducible, larger than usual, tender to touch, very painful, and the percus-

sion note is dull. As a rule, vomiting is an early symptom and one that increases in severity. When the vomiting is an early symptom, it is due to reflex causes, later the vomiting is due to waves of reverse peristalsis, which cause regurgitation. At first the vomitus is from the stomach, next from the upper part of the duodenum and later the vomitus may be fecal in character. Early in the case there may be some temperature, later becoming normal or sub-normal. The pulse is irregular, weak and rapid. As time passes, the pains in the abdomen and hernia become more severe, and collapse frequently follows. When gangrene begins the symptoms become much less severe. There is not so much pain or nausea and vomiting becomes much less. Prostration becomes more marked; the pulse is weaker, more irregular and intermittent; the collapse deepens, and delirium follows.

Treatment—For a reducible umbilical hernia: Prevent constipation. Do not allow patient to take severe exercise or strain, and make patient wear a truss. The radical cure is an operation, as follows: Make an incision around the mass and separate the sac from the superficial tissues, if possible. If this can't be done, open the sac and separate it from the contents, then return the contents to the abdominal cavity. The sac is isolated and removed. The peritoneum is then closed by means of continuous catgut sutures. If possible the recti muscles are to be pulled together to close the opening; then the aponeurosis is closed with continuous cat gut sutures. A few sutures may be put in the subcutaneous fat and then the skin is closed with a continuous suture of silver wire; after which the patient is to be dressed under aseptic precautions.

AIR EMBOLISM.

By William F. Rice,
Junior Medical Student.

Of significant importance to medical men, and to surgeons in particular, is the subject of air embolism, a condition due to the entrance into the venous circulation of a quantity of atmospheric air.

That the forcible injection of air into the veins would cause death was known by observers as early as the sixteenth century, and Ferrier, in 1806, first noted a case where the air entered spontaneously into the venous circulation. Ammussat, in 1839, collected thirty-two cases of air embolism. Of these, nine were proven by autopsy; six, while not confirmed, were undoubted; five cases were doubtful, and the remainder recovered.

Experiments have proven that such animals as the dog and the horse can stand the forcible injection of large amounts of air into the venous circulation without bad effects, if the introduction is made slowly, so it would seem that man is much more susceptible or that the danger has been exaggerated.

Welch intimates that many of the cases reported were not really properly diagnosed, and that the gas found in the heart and vessels after death was not atmospheric air, but gas produced by the Bacillus aerogenes capsulatis, and he has isolated this organism from bodies after death. He insists that in no case is the diagnosis positive unless a bacteriological examination has been made and the absence of this organism noted.

In operations about the upper chest wall and the neck, as in the removal of growths, resection of the clavicle, reduction of shoulder dislocations, etc., the surgeon must exercise the greatest caution, for, as can be seen by the table below, there is a suction in the large veins of this region during inspiration, rendering the patient very susceptible to air embolism in case of injury to one of these vessels.

TABLE OF PRESSURE.

Left innominate vein,	— 0.1 mm. of mercury.
Right jugular vein,	+ 0.2 mm. of mercury.
Right subclavian vein,	— 0.1 mm. of mercury.
Left subclavian vein,	— 0.6 mm. of mercury.
Left jugular vein,	— 0.1 mm. of mercury.

Owing to the peculiar relations of the external jugular vein to the cervical fascia, and of the auxiliary vein to the costo-coracoid membrane, whereby these vessels are prevented from collapsing when injured, they offer favorite sites for the entrance of air. This region has therefore been termed "the danger zone," and surgeons, in operations in this locality, should use precautionary measures not considered necessary elsewhere, for instance, the large veins nearby should be located and either temporarily clamped or ligated and cut proximal to the seat of operation.

Cases of air embolism have been reported in connection with operations on the cranium, the

cranial sinuses offering easy means of ingress for the air, on account of the elevated position of the head during operation, the rigidity of the sinus walls, and the free hemorrhage accompanying such operations.

During pregnancy the uterine veins become enormously dilated, and when the placenta is detached some of the veins may be exposed. If the uterus contracts, and remains so, all right, but, if after contraction the uterus relaxes and the uterine cavity contains air, this may be drawn into the veins by the suction produced. Important factors to be noted here are: The elevation of the pelvis in certain operative positions, the anaemic condition of the patient, and the dilatation of the veins, particularly those of the broad ligament.

Skepticism is shown regarding reported cases of air embolism caused by injection of substances into the uterus for purposes of abortion, the probability being that the gas is formed by an organism as before noted. The same doubt attends the cases of Jurgensen's, where air was supposed to have entered gastric and intestinal veins, but some credence is given Janeway's hypothesis that transitory hemiplegia and other cerebral symptoms, which have been observed to follow washing out the pleural cavity with hydrogen peroxide, were the result of air embolism in the cerebral vessels.

When a vein in the "danger zone" is injured a peculiar hissing, gurgling or sucking sound is heard, which may be loud or scarcely audible, attended usually with bubbles of air in the wound. If the patient be conscious he will utter an anguished cry. There will be symptoms of collapse, face becomes pale and livid, the pupils are dilated and reflexes of the body abolished. On account of the embarrassment of the pulmonary circulation, respiration will be irregular, the pulse accelerated and scarcely perceptible. A churning systolic sound, due to the presence of air in the heart cavities, is heard on auscultation. If the patient is conscious convulsions, more or less severe, may develop.

Various views are entertained as to the cause of death in such cases.

Morgagni held that air bubbles, lodged in small veins, distended and pressed on the nerve centers, and his view has gained some weight from the results of experiments. It has been shown by some that the heart continues to beat for sometime after cessation of life. Air, injected into the carotid arteries, produced death in the same way as when injected into the veins.

Morgagni, in his post-mortem examination, claimed to have found froth in both ventricles, showing, if true, that the air must have passed through the pulmonary capillaries.

While this view as to the cause of death is not now strongly upheld, it is believed by many that cerebral injury is a contributary factor in the fatality.

Others attribute death to a breaking down of the air spaces and the formation of cavities in the lungs, but not much support is given to this theory.

While the cerebral and pulmonary theorists have their supporters, experimental and clinical evidence justify us in believing that the primary cause of death is the effect of the air on the heart. In the tricuspid insufficiency, sudden distention of the ventricles and obstruction of the coronary vessels, there is enough to account for the serious and fatal embarrassment of the heart action.

The anti-mortem diagnosis of air embolism can be made on the symptoms enumerated above, viz., respiratory embarrassment, convulsions, feeble and irregular pulse, characteristic sound on entrance of the air into the vein, and the reflux of blood mixed with air bubbles from the injured vein during systole.

The autopsy shows the right cavities of the heart distended with frothy blood and blood containing air bubbles is found in the pulmonary artery and in the large veins in the vicinity of the heart.

Treatment.—Since it is known that small quantities of air in the veins leads to fatal conditions, care should be exercised in performing operations in the "danger zone." As soon as the injury to the vein is detected pressure should immediately be made upon the proximal portion of the vessel and the wound filled with normal salt solution to prevent ingress of any more air.

Stimulants should be given; nitrite of amyl inhalation acts rapidly on the heart, and by causing rapid dilatation of the vessels aids in relieving the heart. General stimulants, such as strychnine, camphor and alcohol, given hypodermically, aid greatly in relief.

Overdistention of the right ventricle is the main cause of fatality, so measures should be instituted to relieve the same.

Puncture and drainage of the right ventricle can be performed by introducing a needle obliquely, from below upward in the left intercostal space between the fourth and fifth ribs, about one and a half inches from the sternal margin. Senn has also recommended catheterization of the right auricle until all the frothy blood has been withdrawn, this to be replaced with normal salt solution at the temperature of about 120° F.

While both of these proceedings are very radical measures, they are justifiable on account of the high mortality in untreated cases of air embolism.

We are in receipt of the following letter from Dr. G. M. Van Poole, class of 1899, Major, Medical Corps, U. S. Army, stationed at Fort Washington, Md.:

"Fort Washington, Md., April 21, 1913.

"Editor of the Hospital Bulletin,
608 Professional Building,
Baltimore, Md.

"My Dear Sir—I read with pleasure the noble addresses and greetings made by the members at the reception tendered Dr. Fell by the General Alumni Association of the University of Maryland on March 27, 1913. I was particularly interested in what Dr. B. Merrill Hopkinson had to say about successful advertising. I think he sounded the keynote of progress, especially in the medical school rôle.

"In my travels throughout the country I have frequently encountered doctors and others, especially in the Western States, who desired to send their sons to an Eastern college. They know practically nothing about the Eastern schools, so they pick up the first medical journal that comes to hand and look under the heading of some of the Eastern States and select a city. Suppose they select Baltimore; they find several schools advertised, and naturally write to one or all of these for catalogues, from which they select a school.

"The Surgeon-General of the Army furnishes the following journals for the use and benefit of the medical officers in the army: *Journal of the American Medical Association, Medical Record, New York Medical Journal, Annals of Surgery, Boston Medical and Surgical Journal, Journal of the Medical Sciences,* and several others, some foreign. In not one of these leading journals does an advertisement of the University of Maryland appear. Of course, the alumni are going to do all they can for their Alma Mater, but some are scattered at quite a distance, and when they see the other schools advertised so vividly and their own not at all, it is bound to make an impression upon them, and when their sons are to enter a medical school they will undoubtedly write for a catalogue from the school from which they were graduated, but at the same time they will write to the advertising schools, too.

"By advertising I don't mean that it should be done in such a manner as to warrant criticism, but do it judiciously and a sufficient amount to keep abreast of some other schools whose achievements, no matter how small, are known throughout the country. We all know that our dear old University needs no advertising; that her teaching staff can be excelled by none; that the facilities for obtaining practical experience are equal to the best; but, unfortunately, the outside world is not aware of these facts.

"The University of Maryland has always had an enviable record; her sons have reached the zenith in their profession, and now that she and her sister, the Baltimore Medical College, have agreed to join hands in the common cause, they should not forget one of the most essential features in any successful business enterprise, *i. e.*, 'it pays to advertise.' "Fraternally yours,

"G. M. Van Poole,
"Major, Medical Corps, U. S. Army,
"Class of '99."

The following graduates (Baltimore Medical College) hold the following positions in the United States military service:

Major William H. Brooks, U. S. A., class of 1889.

Major Roger Brooke, U. S. A., class of 1900.

First Lieutenant Lloyd A. Kefauver, U. S. A., class of 1906.

First Lieutenant Madison H. Bowman, M. R. C., U. S. A., class of 1892.

Passed Assistant Surgeons, U. S. Navy:

Lieutenant James S. Woodward, class of 1901.

Lieutenant Fletcher H. Brooks, class of 1902.

Lieutenant Clarence E. Strite, class of 1902.

United States Public Health:

Assistant Surgeon H. E. Hasseltine, class of 1904.

THE HOSPITAL BULLETIN

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Editors

Nathan Winslow, M.D. J. M. H. Rowland, M.D.

Baltimore, April 15, 1913.

RETROSPECT, SESSION 1912-13.

Another academic year is rapidly approaching completion, and will soon be numbered with the long line of its predecessors. In what manner does it differ from those that have preceded it, or have any material advances been made? In the first place, an enormous amount of housecleaning has been done; the various buildings have been renovated and put in fine condition, and useful and much-needed equipment has been procured. After many years of solicitation we have finally succeeded in getting smooth asphalt and wooden-block pavements on Lombard and Greene streets, thereby lessening the noise and dust. For this benefit we are largely indebted to Prof. Timothy O. Heatwole, Dean of the Dental Department, and a member of the City Council. The teaching faculty has been reorganized and placed on a university basis; that is, seven full-time teachers have been employed during the session, who have given their whole time to the University, besides several others who were practically full-time instructors. By this change our laboratories are now in charge of men who devote their time to the school, and do not practice medicine for a livelihood. This is a condition that differs largely from that of previous years, and is also a great advance. The students have been more thoroughly trained in the fundamental branches, as well as in practical clinical work. Professor Zueblin has practically revolutionized the medical teaching, and has worked faithfully with the men in the wards and dispensary; and has made them do much microscopical and chemical investigation in the clinical laboratories. He has also established practical bedside examinations of candidates for graduation, in addition to the theoretical written examinations. In surgery much the same methods have been introduced, and the effort has been made to teach by demonstration rather than by didactic means. In some instances proficient students have been allowed to perform minor operations on patients, under the strict tutelage of the instructor, as well as to give anesthetics under supervision. Our superb obstetric clinic affords magnificent opportunities for the practical instruction of senior students, which is fully utilized. We might mention the various special departments in which practical clinical instruction and practice has largely superseded other methods of teaching. We think, therefore, that the work of the session that is closing has been well done; in fact, that it has been better done from the pedagogic standpoint than ever before. After January 1, 1914, a year's work of collegiate grade in chemistry, physics, biology and either German or French will be required in addition to a completed four-years' high-school course for admission to the freshman class, and this will insure a better educated and more competent class of students in the near future. Two events of great importance, both to the medical department and to the university as a whole, have also occurred during the present academic year, and serve to differentiate this session from the others that have preceded it. The first of these events was the election of Thomas Fell, L.L.D., D.C.L., as Provost of the University of Maryland. Dr. Fell is a distinguished educator and an able administrator, and from that viewpoint his election is satisfactory; but he is the first Provost under salary and exercising actual executive functions as the head of the institution, and we believe his inauguration means a new era in the history of the University. We now have a head, with executive offices at the University, and the necessary equipment for efficient work. The other event is the merging of the medical and dental schools of the Baltimore Medical College with those of the University. By this means the number of competing schools is diminished, additional equipment secured, another large hospital brought under our clinical control, and a number of able teachers added to our corps. The large

and loyal alumni body of the Baltimore Medical College has been made eligible to election in the General Alumni Association of the University, and will doubtless prove loyal and useful members. In looking backward we have good reason for felicitating ourselves on the progress made, and of entertaining the belief that we have entered upon the road that leads to success.

THE CLASS OF 1873.

The class of 1873 consisted of 46 members, of whom only 19 survive at this time. While most of its members have been prominent and successful physicians in their respective localities, the class is remarkable for the fact that three of its members have been professors and regents of the University and several others have held chairs in other medical schools.

Dr. J. Edwin Michael was successively professor of anatomy, and of obstetrics, and held the latter chair at the time of his death in 1895.

Dr. Randolph Winslow was elected to the chair of anatomy and clinical surgery in 1891, and succeeded Dr. L. M. Tiffany in the chair of surgery in 1902.

Dr. Thomas A. Ashby was elected professor of diseases of women in 1897.

Dr. Geo. H. Rohe was for many years a professor in the College of Physicians and Surgeons of this city, and was the head of the Spring Grove Hospital for the Insane, and subsequently of the second hospital for the insane at Springfield. He died in 1899.

Dr. T. Morris Murray held a chair in Washington, D. C., and Dr. Virginius W. Gayle one in Kansas City, Mo.

Four members of this class have also been elected President of the Medical and Chirurgical Faculty of Maryland—Dr. Ashby in 1890, Dr. Rohe in 1893, Dr. Michael in 1895, and Dr. Winslow in 1913, to assume office in 1914.

AN EXAMPLE TO BE EMULATED.

The Medical College of South Carolina has had quite a close connection with the University of Maryland. In 1831 Dr. Eli Giddings, a man of great erudition, was called from Charleston, S. C., to the professorship of anatomy and physiology in the University, but in 1837 he returned to Charleston and was made professor of surgery in the Medical College of South Carolina, with which institution he continued to be associated for 40 years. He became the most prominent physician in his city and State, and enjoyed a national reputation. In 1868 Dr. Julian J. Chisolm, professor of surgery in the Medical School at Charleston, was elected to a chair in this University, and subsequently was professor of eye and ear diseases until his resignation in 1895. He was the most instructive teacher that the writer has ever listened to, and he became a man of great distinction in his specialty. In 1869 Dr. Francis T. Miles, a former professor of anatomy in the Medical College of South Carolina, was elected professor of anatomy, and later professor of physiology in this University. He was a person of rare charm of speech and manner, and was a great ornament and asset until his death in 1903. We have, therefore, been indebted to this South Carolina college for three of our most distinguished professors, whose memories cling like a fragrant perfume to our halls. This college has not been able to keep up with the advance of medicine, and was placed in Class C by the Council on Medical Education of the American Medical Association. Recently it has been absorbed by the State University as an integral factor, and by a whirlwind campaign was able to raise $76,000 in 10 days from the citizens of Charleston. We have derived great benefit from the teachers who were trained in this school, and we rejoice in its good fortune and hope that it is on the road to prosperity. But the lesson that we especially desire to impress upon our rulers is that we, in a similar manner, must make a vigorous demand upon the citizens of Baltimore and Maryland if we expect to get the endowment, without which we shall not be able to exist.

THE PATHOLOGICAL ENDOWMENT FUND.

This fund grows slowly. Small favors are thankfully received and larger ones in proportion. If one adds up the column of figures of the contributions by classes, it will not be found to correspond with the sum total as reported each month. It is not intended to do so. There are other contributions by persons who are not graduates. The total is correct, however.

CONTRIBUTIONS BY CLASSES.

1848	$50 00
1904	20 00
1908	10 00

1871	35 00
1872	81 84
1873	491 83
1874	5 00
1875	5 00
1876	115 00
1877	10 00
1880	5 00
1881	252 00
1882	310 00
1883	40 00
1884	40 00
1885	235 00
1886	100 00
1888	50 00
1889	100 00
1890	175 00
1892	150 00
1893	40 00
1894	135 00
1895	155 00
1896	52 00
1897	80 00
1898	115 00
1899	105 00
1900	220 00
1901	270 00
1902	330 00
1903	340 00
1904	135 00
1905	245 00
1906	195 00
1907	120 00
1908	45 00
1909	15 00
1910	75 00
1911 Terra Mariae	3 50
1912 Club Latino Americano	25 00
Total subscriptions to May 1, 1913	$10,542 17

NEW SUBSCRIPTIONS IN APRIL.

Harry A. Cotton, 1899	$50 00
Robt. L. Mitchell, 1905	25 00
Total	$75 00

ITEMS

The following completes the list of graduates of the University Hospital Training School for Nurses, a partial list of which appeared in March BULLETIN:

CLASS OF 1906.

Mrs. Ethel P. Clarke, University Hospital, Baltimore.

Sara M. A. Sanderson, Baltimore.

Clara E. Query, Baltimore.

Sara W. Cunningham (Mrs. Arthur W. Morse, U. S. A.).

Katharine Landwehr (Mrs. Charles Lemley), West Virginia.

Nellie H. Carter, Chatham, Va.

C. M. Phillips (Mrs. Greufell).

A. E. Chapman (Mrs. J. E. Wright), Easton, Md.

Mary C. Ellicott (married).

Leonore G. Doyle (Mrs. W. W. White), Baltimore.

Mrs. Georgia A. Truitt (dead).

Miriam L. Jessups (Mrs. F. B. Hines), Chestertown, Md.

Margaret E. Lawrence, Baltimore.

CLASS OF 1907.

Amy B. Tongue, Mt. Vernon, N. Y.

M. W. Pue.

Rosamond Minniss, Walker Hospital, Wilmington, N. C.

Naomi V. Hissey, Baltimore.

Nannie L. Brian, Rocky Mount, N. C.

Grace I. Bay, Baltimore.

Lula E. Minor, Baltimore.

Mary E. Grimes, Baltimore.

Alice F. Bell, Annapolis, Md.

Isabella Griffith, Baltimore.

Esther E. Brewington, Walker Hospital, Wilmington, N. C.

Jane T. Haydon, Baltimore.

Ella B. Ogburn.

Nancy M. McNabb, Baltimore.

Clara B. Robinson, Baltimore.

Corrie F. Peyton, Baltimore.

Jennie D. Barber, Baltimore.

CLASS OF 1908.

Martha R. Hamlin.

Clyde C. Dawson (Mrs. F. S. Lynn), 1619 St. Paul street.

Henrietta U. Gourley, 2128 St. Paul street, Baltimore.
Augusta C. Russell, Jacksonville, Fla.
Ethel P. Schull.
A. R. Cunningham.
Minnie B. Anderson (Mrs. R. B. Hayes), Fayetteville, N. C.
Mary Gavin, The Walbert, Baltimore.
S. A. Howstrauser, Baltimore.
Rose Wilson.
Charlotte A. Cox, Baltimore.
Mary E. Wright (Mrs. G. H. Richards), Port Deposit, Md.
Lulu A. Price.
Maude F. Smith (Mrs. S. R. Cornelius), Baltimore.
Mary V. Hamlin, Danville, Va.
Harriet J. Parsons, The Walbert, Baltimore.

CLASS OF 1909.

Catherine M. Dukes, 1425 John street.
Mrs. Anna M. Green.
Laura S. Chapline, 2318 North Charles street.
Louise D. Pue, Baltimore.
Grace S. Tull, 1425 John street.
Annie C. Wham, University Hospital.
Eva S. Chapline, 2318 North Charles street.
Beulah O. Hall, Gainesville, Fla.
Elizabeth M. Getzendaner, Guilford Apartments, Baltimore.
Emily L. Ely, 2034 Druid Hill avenue.
Lucy B. Squires, Jacksonville, Fla.
Gertrude H. Tews (Mrs. L. G. Cole), Jessups, Maryland.
Helen M. Robey, 515 Cecil avenue.
Blanche Almond, Virginia.
Lettie B. Carter, University Hospital.
Mary B. Saulsbury (Mrs. J. H. Bay), Havre de Grace, Md.
Vera Wright, Navy Nurses Corps, Washington, D. C.

CLASS OF 1910.

Ellen C. Israel, 1403 Madison avenue.
Amelia A. Strohm, Brooklyn, N. Y.
Gertrude A. Garrison (Mrs. R. D. McMillan), Red Springs, N. C.
Adele D. Barrett, Georgia.
Margaret M. Taylor, Baltimore.
Florence A. King, Fayetteville, N. C.
Mary V. Kimmel, Kernan Hospital.
Sarah A. Lee, Baltimore.
Sarah L. Long, Baltimore.
Lula C. Price, Baltimore.
Cora N. Burton, 640 West North avenue.
Virginia O. McKay, Baltimore.
Marie B. Murchison, Baltimore.
Annie M. Drye, Baltimore.
Martha V. Edmunds (Mrs. Dwight G. Rivers), Fort White, Fla.
Pauline B. Pleasants, Baltimore.
Mae C. Wiggin, U. S. N., Brooklyn Navy-yard.
Lucy B. Barber, Baltimore.
Agnes Holland, Baltimore.
M. Louise Gephart, 2410 Maryland avenue, Baltimore.
Fannie Meredith, Baltimore.

CLASS OF 1911.

Annie S. Grubb.
George A. Hulton.
Frances W. Sprecher, 1403 Madison avenue.
Elizabeth C. Patterson, Baltimore.
Barbara Stouffer, Hampton Court Apartments, Baltimore.
Mary G. Brady, 640 West North avenue, Baltimore.
Nellie E. Curtiss, 1403 Madison avenue, Baltimore.
Eva Robinson, Baltimore.
Marvel Scarff (Mrs. J. H. Von Dreele), 846 West 36th street, Baltimore.
Stella W. Ricketts, Baltimore.
Naomi Helland, 1403 Madison avenue, Baltimore.
Ivy I. Kinney (Mrs. Judson Hair), Coketown, West Virginia.
Alva Williams, Hampton Court Apartments, Baltimore.
Ruth E. Berlin (Mrs. Chipman), Detroit, Mich.
Mary E. Sullivan, University Hospital.
Jennie R. Garner, University Hospital.

CLASS OF 1912.

Mattie Coale, Baltimore.
Agnes Lynch (Mrs.), Virginia.
Marion Smith.
Alice Wells.
Lucy Harvey, 1305 Madison avenue.
Juliette Miles, City.
Eulalia Cox.
Bernice Conner, 640 West North avenue.
Lena Stouffer, Hampton Court Apartments.
Mary Steiner, Annapolis.
Eliza Ridgeley, Baltimore.
Ethel Logue, Baltimore.

Lillian Blake, 1403 Madisonavenue, Baltimore.
Blanche Prince.
Ethel Dawson, University Hospital.
Lucy Lilly, 1115 Madison avenue, Baltimore.

CLASS OF 1913.

Dorothy Henrietta Patterson, Pennsylvania.
Martha Misikofski, Maryland.
Willie Brown Hull, Virginia.
Evelyn Houston Chase, Virginia.
Edith Mildred Brownell, Rhode Island.
Adelaide Caroline Coward, North Carolina.
Sophia Frances Hessler, Maryland.
Golda Gleneith Price, Virginia.
Mary Rennie, Maryland.
Elva Lydia Dean, Maryland.
Mary Myrtle Selby, Maryland.
Margaret Gertrude Laws, Maryland.
Anna Elizabeth Butts, Maryland.
Volina Maybell Rutherford, Virginia.
Mary Ann Rutherford, Virginia.
Pearl Levora Rush, Maryland.
Katherine Veronica Shea, Massachusetts.
Natalie Isabel McCann, Maryland.
Katherine Woodall Welch, Maryland.
Edith Dent, Washington.

Dr. Lemuel Offutt, class of 1876, of Greensburg, Pa., recently spent a few days in Baltimore. Dr. Offutt enjoys a large and lucrative practice, is a man of immense importance in his community and is held in the highest esteem by all with whom he comes in contact. He has a wide medical experience, both practical and theoretical, which he constantly puts into use both in his own practice and consultation with his brother-physicians.

Dr. Charles C. Croushore, class of 1905, is also located at Greensburg, Pa.

Dr. A. P. Herring, Baltimore Medical College, class of 1896, secretary to the State Board of Lunacy, notified Superintendent Gregg of Montevue Hospital that all female negro patients from other counties would be removed to the State institution at Crownsville May 10, the white patients from other counties having previously been removed. In a short time only Frederick county patients will be left at Montevue.

Dr. Thomas Fell, provost of the University of Maryland and president of St. John's College, recently attended the sessions of the Southern Educational Conference in Richmond, Va. Dr. Fell represented both institutions over which he presides.

Dr. G. Carville Mace, class of 1897, who has been ill at the University Hospital, is much improved.

Miss Jennie Garner, head nurse of the maternity ward, University Hospital, who has been ill with an infected foot, is much improved.

Mrs. Louis B. Henkel, wife of Dr. Louis B. Henkel, Jr., class of 1903, of Annapolis, Md., recently underwent an operation at the University Hospital.

Dr. A. Duvall Atkinson, class of 1894, and Mrs. Atkinson have left for Norfolk, Va., where Dr. Atkinson is recuperating from a recent attack of pleurisy.

Dr. W. Milton Lewis, class of 1888, and Mrs. Lewis of 1400 Linden avenue have opened their country home at Lutherville, Md.

William Culbert Lyon, class of 1907, formerly of Baltimore, Md., is now an Assistant Surgeon in the U. S. Navy, stationed at Richmond, Va., on recruiting duty. We are glad to hear of Dr. Lyon's success, and trust that his work will be most congenial to him.

In response to a request, we are glad to give the list of the class of 1903, as follows:

Willis Alston, Jr., Littleton, N. C.
John Frazee Armentrout, Staunton, Va.
Howard Elmer Ashbury, 1017 Cathedral street, Baltimore.
Guy Philip Asper, Chambersburg, Pa.
Augustus A. Babione, Luckey, O.
Albert Lee Barrow, Konnorock, Va.
Monsel Ray Bell, Keyser, W. Va.
Josiah Slicer Bowen, Mt. Washington, Md.
Horace Russell Boyer, Oakland, Cal.
Hugh Warren Brent, 906 N. Calvert street, Baltimore.
Beverly Waugh Briscoe, Accident, Md.

William Irvin Buppert.

J. Walter Burch (dead).

Joseph Henry Cahoon, 253 W. 22d street, New York.

John William Carroll, Wallace, N. C.

Henry Poindexter Carter, M. C. 1st Lt. U. S. A., Fort D. A. Russell, Wyoming.

Daniel David Coffey, 1347 Noble street, Chicago, Ill.

Frank Scott Cooper, Roanoke, Va.

Naim Suleiman Cotran, medical officer in Egyptian Army.

William Wilhelm Craven, Huntsville, N. C.

Robert Orr Crist (died May, 1907).

Richard H. V. Dann, Elmira, N. Y.

Joseph L. De Cormis, Accomac, Va.

Harry Cramer Donahoo.

Byron W. Eakin, Carlisle, W. Va.

Albert D. Edwards, Roanoke, Va.

Lester J. Efird, Tampa, Fla.

Joseph Gamewell Evans.

Walter Hollis Everhart, Newton, N. C.

Robert Waldorf Fisher, Morgantown, W. Va.

Willis Bryan Fitch, St. Johnsbury, Vt.

Manuel Fossas, Manual, Bayaman, Porto Rico.

Bernard S. French, 1514 Hollins street, Baltimore.

Charles Wofford Gentry, Greenville, S. C.

Harold H. Hartley, Jr.

Louis Barnard Henkel, Jr., Annapolis, Md.

Henry Marvin Hodgson, Lonaconing, Md.

Howard Steele Holloway.

Arthur Ralph Hunter, Simpsonville, S. C.

J. Howard Iglehart, 539 N. Carrollton avenue, Baltimore.

Rollin Jefferson, Jr., 246 Hyde Park avenue, Tampa, Fla

Harry Otis Johnson, Machias, Maine.

Howard Wilbur Jones, 222 Augusta ave., Baltimore.

William Merritt Jones, Jr., High Point, N. C.

Shadid Abraham Khuzami, medical officer in Egyptian Army.

George S. M. Kieffer, Morrell Park, Md.

Samuel J. King, Grand Junction, Tulsa county, Colorado.

William Elmo Kurtz (died May, 1907).

Harry Allen Lakin, 714 W. 8th street, Erie, Pa.

Albert Lafayette Levy, 1229 E. Madison street, Baltimore.

Arthur F. Lindley.

W. Clinton Linville, Winston-Salem, N. C.

Richard Nuckolls Littlejohn, Jr.

George Carroll Lockard, 1631 W. Lafayette avenue.

Evel H. Lyon, Rougemont, N. C.

Howard J. Maldeis, Kate avenue, Arlington.

Thomas Allen Mann, 911 N. Mangum street, Durham, N. C.

S. Dace McPherson, Durham, N. C.

Herbert Meyers, Confluence, Somerset county, Pennsylvania.

J. Hernando Minor (unknown).

Fred Clifton Moor, Tallahassee, Lean county, Florida.

Eugene Hagan Mullan, Immigration Service, Ellis Island, N, Y.

James Arthur Norton, Conway, Harry county, North Carolina.

Thomas J. O'Donnell, 405 Warren avenue, Baltimore.

John T. O'Mara, 1042 Edmondson avenue, Baltimore.

Charles Augustus Overman (not in practice), Baltimore.

William J. Pabst.

Ector C. Patterson.

Robert William Petrie, Charlotte, N. C.

Joseph Battle Philips, Jr., (unknown).

Charles Richardson, Belair, Harford county, Maryland.

Joseph Potter Riggs.

Walter Daniel Riordan, 187 Newbury street, Lawrence, Mass.

Arnold M. Rosett, 2221 Fitzwater street, Philadelphia, Pa.

Joshua Rosett, 1503 E. Baltimore street, Baltimore.

Leland Blackwood Salters, Blenheim, S. C.

Clifford T. W. Sappington, Libertytown, Maryland.

Walter Wesley Sawyer, Shiloh, N. C.

George R. Sledge, Parksby, Va.

Laomi J. Smith.

L. Nathaniel Spengler, Donalsonville, Ga.

Charles Edward Terry, 2601 St. John's avenue, Jacksonville, Fla.

Guy Franklin Thigpen.

William Ferguson Torbitt, Bennett, Neb.

Felix Villamill, Goshen, N. Y.

Daniel Alvey Watkins, 115 N. Potomac avenue, Hagerstown, Md.

Frank Watkins Weed, Fort Totten, N. Y.

George Frederick Whelpley, 420 Massachusetts avenue, Boston, Mass.

Harry L. Whittle, Waverly, Md.

Frederick J. Wilkins, 1507 N. Broadway, Baltimore.

Albert Livingston Wilkinson, Belair road and Cole avenue Baltimore.

Charles S. Williamson.

Mark Stanley Wilson, Marlington, W. Va

Richard Eugene Windley, Swanquarter, N. C.

Fairfax G. Wright, 153 S. Main street, Chambersburg, Pa.

Calvin Todd Young, Plant City, Fla.

We will be very glad to receive information regarding the missing alumni.

The following have been elected officers of the Adjunct Faculty for the ensuing year:

President—Nathan Winslow, class of 1901.

Vice-President—E. H. Kloman, class of 1910.

Secretary-Treasurer—A. H. Carroll, class of 1907.

Executive Committee—J. W. Holland, class of 1896; R. L. Mitchell, class of 1905, and R. P. Bay, class of 1905.

UNDERGRADUATE NOTES

Under the Supervision of E. Kilbourn Tullidge.

Mr. L. B. Crumrine, clinical assistant in the University Hospital, recently underwent an operation for deviated septum and is now convalescing.

Dr. Irving J. Spear held his final examination for the senior class on diseases of the nervous system in Davidge Hall, May 1. The examination was exceptionally fair and should have been passed by all.

The annual banquet of the Sigma Tau Chapter of the Theta Nu Epsilon Society was held at the Rennert Hotel on Friday, April 25. Prof. A. M. Shipley acted as toastmaster. The elaborate menu and program of splendid toasts were proof that all enjoyed the evening.

The Kappa Psi Fraternity is preparing to keep their house, 242 West Hoffman street, open during the summer months.

The Phi Sigma Kappa Fraternity have renovated their house on Mt. Royal avenue, and have decided to keep it open during the summer.

The following members of the senior class took the examination on Saturday, April 26, for interneship at St. Joseph's Hospital:

Frank W. Gemmill, Frederick L. McDaniel, S. A. Alexander, William H. Scruggs, Jr.

Prof. Gideon Timberlake held his final examination in Davidge Hall for the senior medical class in genito-urinary diseases on May 9.

The Latin-American Club held its annual banquet at the Rennert Hotel Saturday evening, April 26. A feature of the evening was the good fellowship that prevailed. President J. M. Buch acted as toastmaster and proved an exceptionally good one, according to all reports. Speeches were made by H. M. Perez and J. Reinicke, class of 1913.

The Undergraduate Department of the BULLETIN wishes the club great success in all its future undertakings.

Messrs. R. B. Hill and N. B. Hendrix of the sophomore medical class have recently recovered from severe attacks of the measles and are now preparing for their final examination.

Mr. Louis A. Birely of the sophomore medical class has recently returned from a week's visit to Lynchburg, Va.

The Nu Sigma Nu Fraternity has again decided to keep its home open during the summer.

Midshipman Karl B. Wilson, United States Navy, Annapolis, Md., was the week-end guest of his brothers, Messrs. B. L. and F. W. Wilson of the sophomore and junior classes, respectively, at 618 West Lombard street.

Mr. H. G. Stoneham has received a communication from Dr. Mead S. Brent, assistant superintendent Central State Hospital, Petersburg, Va., offering him an interneship in that hospital.

Dr. Hiram Woods held his final examination on diseases of the ear Monday, May 5, at 8 o'clock P. M., in Davidge Hall.

The annual Adjunct Faculty smoker given the members of the senior class, Saturday evening, May 3, 1913, was a thoroughly enjoyable affair, and the hospitality shown the class was much appreciated.

Mr. William H. Scruggs, Jr., of the senior medical class, has accepted an appointment offered him by Dr. Gordon Wilson as resident physician of the State Tubercular Hospital at Sabillasville, Maryland.

The practical examinations in medicine given by Professor Zueblin have proved of exceptional benefit to the members of the senior medical class, not only brushing them up in theory and practice, but refreshing their memories in the work gone over in the junior year.

The Psi Omega Fraternity gave a very successful and delightful dance to the graduating class Friday evening, April 25.

The undergraduate department of the THE BULLETIN wishes to thank those who, by their subscriptions and contributions, have helped to make this year's publication a success, and who we trust will continue their interest in the paper and thus keep in touch with their Alma Mater. It also wishes God-speed to those who will leave this year for that long, rough, winding road that leads to success, and trusts that they will ever remember and look back upon those happy days spent in the University of Maryland with fond recollections.

BIRTHS

To Dr. Harry D. McCarty, class of 1905, of 613 Park avenue, and Mrs. McCarty, April 25, a daughter, Mary DuBois.

MARRIAGES

Miss Annie W. King, class of 1903, University Training School, of Annapolis, Md., to Mr. Frederick Seiling of Elkridge, Md., April 30, 1913. They will reside at Elkridge.

Dr. Clifton N. Devilbiss, class of 1910, of Laytonsville, Md., formerly assistant resident surgeon in the University Hospital, was married to Miss Adelaide Hoffman, formerly a pupil nurse in the University Hospital Training School for Nurses. After a short wedding trip Dr. and Mrs. Devilbiss will reside in Laytonsville, where Dr. Devilbiss is engaged in the practice of his profession. Both Dr. and Mrs. Devilbiss were very popular during their residence in the University Hospital, and their many friends desire to take this opportunity to wish them much happiness.

DEATHS

Dr. Marcellus B. Shupe, class of 1885, died at his home at Connellsville, Pa., March 22, 1913, from the result of injuries received in a runaway accident a year before, aged 60 years. Dr. Shupe was a member of the Medical Society of the State of Pennsylvania, local surgeon for the Baltimore & Ohio Railroad at Connellsville, a member of the Board of Health, and for three terms a member and once president of the local School Board.

Dr. James Edwin Harris, class of 1886, died in a sanitarium in Baltimore April 8 from cerebral hemorrhage, aged 49 years. Dr. Harris for about 15 years after his graduation in medicine and dentistry was a practitioner in London, England; thereafter for six years a resident of Cape Town, Cape of Good Hope, South Africa, and for the last six years a resident of Baltimore.

Dr. Charles F. Nichols, class of 1887, died at his home in Vienna, N. Y., April 4, 1913, aged 47 years.

Dr. Joel B. Yingling, class of 1878, died at his home, 705 Dolphin street, April 14, 1913, aged 65 years.

Dr. Hiram H. Gunby, class of 1855, died at his home in Crisfield, Md., April 8, 1913, after a brief illness of pneumonia. He was born in Somerset county, Maryland, April 13, 1832. He was the son of Elisha Gunby and Milcah Coulbourne. He left three sons—Edward R. Gunby of Tampa, Fla.; A. B. Gunby of Boston, Mass., and David N. Gunby of Arizona. Dr. Gunby was a nephew of a distinguished officer of the American Revolution, Col. John Gunby. He was a member of the Maryland Legislature in 1862; a man of splendid physique, tall, dignified bearing, with graceful and engaging manners; a true type of

the old-school gentleman and physician, which are rarely seen. He was a member of the Somerset County Medical Society and an ex-member of the American Medical Association.

BOOK REVIEWS

International Clinics. Vol. I. Twenty-third Series. Philadelphia and London: J. B. Lippincott Company. 1913.

As acute anterior poliomyelitis has become so prevalent and so widespread, though no specific treatment has been devised, yet as the ravages of the disease are so terrible, it is indeed timely to have an article on its treatment. The author, Dr. George E. Malsbary of Los Angeles, Cal., rightly says as there is no satisfactory specific therapy, the treatment of poliomyelitis consists largely in the prevention of the spread of the disease, ridding the body as far as possible of the virus that causes the disease, the application of such general and local measures as will tend to lessen the undesirable early pathological changes, the relief of dangerous and unpleasant symptoms, and later the relief or correction of the sequela, especially paralysis and deformity. In preventing the spread of poliomyelitis the writer advises strict quarantine, the liberal use of screens and the destruction of the breeding places of flies. A rational attempt at quarantine, according to the author, implies an exact knowledge of the virus, which, according to the various theories so far propounded, he proceeds to enumerate (the article was published before Flexner's announcement of the discovery of the specific bacterium). He states the resistance of the virus enables it to be carried by both active and passive carriers, notably by clothing, bedding, domestic pets and insects. It is not readily destroyed by dessication, so that possibly it may be disseminated by dust. Taking these facts into consideration, one easily comprehends the necessity for the strictest quarantine. The body may be rid of the virus by agents which may be used in the respiratory tract without damage, as it is here that the poison finds chief lodgment. Among these agents the author recommends especially hydrogen peroxide and menthol, the peroxide being in the strength of 2 per cent. and the menthol 0.5 per cent. He also recommends early in the disease catharsis as of value in getting rid of the virus. Hexamethylenetetramine can be employed advantageously, but is not a specific. Saturation with this drug delayed the onset of the disease in monkeys. The rest of the article he gives over to the treatment of symptoms and the prevention and treatment of paralysis and deformity and a summary of his conclusions. It is a well written article and alone worth the price of the book. The rest of the volume is a live wire, full of interesting articles by well-known writers, any and all of which should be of interest not only to the general practitioner, but in many instances to the specialist.

Private Duty Nursing. By Katherine DeWitt, R.N., Graduate of Mt. Holyoke Seminary and of the Illinois Training School for Nurses; Assistant Editor of the *American Journal of Nursing*. Cloth, $1.50 net. 1913. Philadelphia and London: J. B. Lippincott Company.

Nursing, like medicine, has become so specialized that no one book can cover its many activities. What is necessary for the hospital nurse to know is not germane to the private duty nurse. Therefore books on the various aspects are gradually coming into existence. One of which is the volume before us. This book endeavors to point out to the new graduate, and, in fact, even the old graduate, the many pitfalls befalling the nursing profession; to show how to avoid unpleasant experiences and how to put into practice the theoretical and practical knowledge learned while in training. A right beginning to the private duty nurse means success, a wrong, perhaps, dismal failure. If the graduate starts on the right tack she generally succeeds, but if on the wrong, whereas she may ultimately be able to overcome the handicap, yet her experience will be bitter and dearly paid for. The book is full of good common-sense and advice which have been gained by the author only after an experience of sixteen years. She especially calls to attention the well-known fact that every time a nurse is called into a family her actions and behavior are critically watched, and the whole profession judged thereby. The book also emphasizes the educational feature of the nurse's calling. One has been prone to look upon the institutional nurse alone as an educator, overlooking this essential office in the outside nurse. Every nurse should possess the book and keep it at hand, so that during any spare moments she may consult it, as it is full of useful suggestions.

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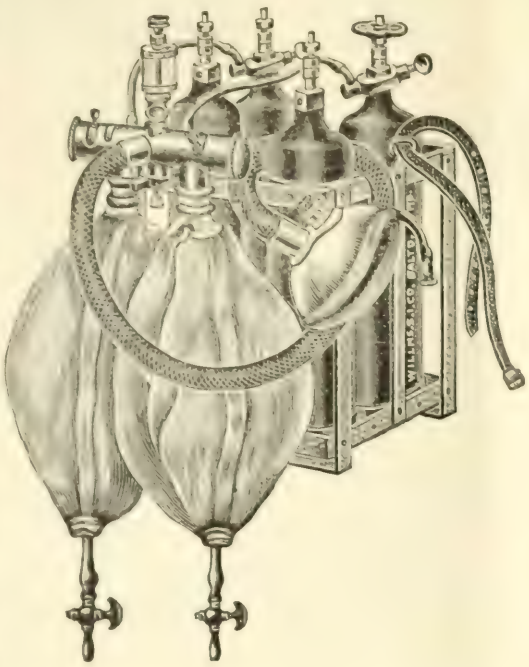

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